protein that is prominent in kidney ducts and zebrafish laterality organ, the Kupffer's vesicle (KV). Furthermore, endogenous Plac8.1 concentrated at apical membranes where cilia were rooted, thus indicating a role in cilia regulation. Indeed, plac8.1 loss-of-function zebrafish embryos manifested a set of morphological defects consist with cilia defects: ventrally curved body axis, left/right asymmetry defects, and kidney cysts. Also in those embryos, motile cilia number in the KV was significantly reduced, and cilia in kidney ducts were abnormally curved and displayed membrane defects around the ciliary axonemes. Besides the morphological defects, zebrafish embryos with reduced Plac8.1 also showed impaired motile cilia beating. Finally, to investigate the molecular mechanism of how Plac8.1 regulates cilia morphogenesis and activity, we are studying potential interactions between Plac8.1 and Cops proteins, the integral components of the ubiquitination regulating complex CSN. Thus, this work reveals a novel component regulating motile cilia, and could implicate ubiquitination in motile cilia regulation in the zebrafish.

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Program/Abstract # 157
Automated training and quantitative behavior analyses of molecularly-tractable model organisms
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Is there a causal link between human handedness and cognitive function? While a number of studies have explored lateralized behaviors (e.g., eye preference) in model organisms whose left–right axial patterning was perturbed, it is largely unknown whether the consistent left–right asymmetry of the heart and viscera has implications for non-lateralized cognitive functions such as learning and memory. The much higher presence of left-handed individuals within both, genius and mental illness-affected populations, remains unexplained. Advances in this area require the synthesis of multiple disciplines, from molecular genetics of processes that give rise to brain abnormalities, to neurological and physiological techniques needed to measure brain function, and finally to behavioral expertise to quantitatively evaluate cognitive performance. Modern molecular techniques avail researchers of the ability to specifically randomize laterality in model organisms, while machine vision technology now allows behavioral assays to be automated, reducing bias and greatly increasing the throughput of learning trials. Here, we report the design and implementation of a custom platform capable of autonomously observing, training, and evaluating memory in *Xenopus* and zebrafish. This system is applicable to many small aquatic model species, and is unique in its ability to provide real-time feedback to animal subjects independently, thus enabling parallelized studies of instrumental conditioning. We used this behavior platform to record baseline metrics of behavior, learning, and memory in *Xenopus laevis* tadpoles. In addition, we have initiated studies comparing the behaviors of wild type individuals to those of tadpoles with induced laterality randomizations or reversals. More broadly, we expect this system to greatly lower the barrier of including behavioral endpoints in pharmacology, toxicology, developmental biology, and neuroscience studies. Automated analysis of cognitive performance will shed light not only on the fascinating link between the asymmetry of body and brain, but will also advance systems biology in enabling studies that span from genes to body structure to behavior and thought.

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Program/Abstract # 158
Critical functions of myocardial Mycn in the developing mouse heart
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Congenital heart diseases (CHDs) are the leading cause of infant morbidity/mortality, and yet the underlying molecular/genetic mechanisms remain poorly understood. We previously identified Mycn as a direct target of the BMP signaling pathway in developing hearts. Consistent with our mouse study, haploinsufficiency for MYCN causes Feingold syndrome, a developmental disease characterized in part by heart defects. We test the hypothesis that myocardial Mycn is essential for cardiomyogenesis through a conditional gene inactivation approach. Loss of myocardial Mycn caused embryonic lethality at midgestation. Mutants displayed severe hypoplastic myocardial walls, which is caused by both decreased cell proliferation and reduced cell size, but not by increased cell death. Expression of cell cycle regulatory genes including Cyclin D1, D2 and Id2 was reduced in mutants. Furthermore, Mycn promotes cell growth through upregulating p70SK expression. Deletion of Mycn led to incomplete trabeculation, resembling mouse models with disruption in the Nrg-1/EphbB pathway. Treating embryonic hearts with an Ephrin-specific blocker severely reduced Mycn expression, suggesting that Mycn is a key target of Ephrin signaling in promoting trabeculation. Deletion of Mycn did not lead to pre-maturation of embryonic cardiomyocytes as evidenced from examining embryonic/adult specific cardiomyocyte markers. In conclusion, our study reveals Mycn as a key transcription factor mediating activities of multiple signaling pathways in promoting cardiomyocyte proliferation and myocardial wall morphogenesis. This information contributes to our better understanding of the molecular mechanisms underlying heart development and CHDs.

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Program/Abstract # 159
Not just inductive: A critical mechanical role for the endoderm during early cardiogenesis
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The primitive heart tube (HT) is the first mechanically functioning organ to form during development. During gastrulation, the cardiac progenitors take up residence in the lateral plate mesoderm but remain in close contact with the underlying endoderm. In amniotes, these bilateral heart fields are initially organized as a pair of flat epithelia that move toward the embryonic midline and fuse above the anterior intestinal portal (AIP) to form the straight HT. This medial motion is typically attributed to active mesodermal migration over the underlying endoderm. In this view, the endoderm's role is two-fold: to serve as a mechanically passive substrate for the crawling mesoderm and to secrete various growth factors necessary for cardiac specification and differentiation. In this work, using both computational modeling and experiments on chick embryos, we present evidence for an active mechanical role for the endoderm during HT formation. Label-tracking experiments suggested that active endodermal shortening around the AIP accounts for most of the heart field motion toward the midline. The myosin II inhibitor, blebbistatin, arrested this shortening and (in many cases) resulted in cardia bifida — thus indicating a role for cytoskeletal contraction. Also, microindentation tests before and after the application of blebbistatin